The Central African Journal of Medicine

Editor:
MICHAEL GELFAND, C.B.E., M.D., F.R.C.P.

Assistant Editor:
JOSEPH RITCHKEN, M.D.

Volume Eighteen

JANUARY - DECEMBER

1972

SALISBURY : RHODESIA

Australia Antigen — Rhodesia

2. SURVEY OF URBAN BLOOD DONORS AND RURAL POPULATIONS

BY

J. G. CRUICKSHANK

AND

R. SWANEPOEL,

Department of Medical Microbiology, University of Rhodesia,

R. F. LOWE,

T. ROBERTSON,

AND

H. MOORE,

Salisbury and District Blood Transfusion Service.

INTRODUCTION

A recent pilot survey indicated that there was a high incidence of Australia antigen (Aul) in the serum of African patients with hepatitis in Rhodesia (Cruickshank and Swanepoel, 1971). Elsewhere in Southern Africa the antigen was demonstrated in 3,4 per cent. of a random sample of an apparently normal African population (Grobbelaar, 1970). An incidence as high as this implies a real risk in circumstances where blood or blood products are handled or administered as in transfusion, renal dialysis, surgical and haematology units.

Consequently a more comprehensive survey was undertaken to determine the general prevalence of the antigen in Rhodesia by testing African and European blood donors as examples of urban populations and, in contrast, certain African tribal groups as examples of rural populations. The results are presented here together with the findings in cases of hepatitis and leprosy which have come to hand subsequent to the pilot survey.

MATERIALS AND METHODS

Urban blood donors

Serum samples were obtained from all donors bled in Salisbury over a four-month period. The African donors were almost exclusively young men apparently in good health.

African donors were generally bled in groups and were drawn from individual industrial firms, the labour exchange, prisons, police, air force, secondary schools, training colleges and the university. A few donors from non-defined sources were bled at community centres and Red Cross clinics.

The European donors were drawn from a rota of individuals.

For comparative purposes the Bulawayo and District Blood Transfusion Service supplied three batches of serum samples from unselected African donors drawn from sources similar to those in Salisbury.

Rural populations

The screening of rural African populations was opportunist, the blood samples being collected from three areas in the course of other surveys:—

(a) Randomly selected men, women and children were bled in villages at Chisunga and Chitsungo in the Dande, Angwa and Hunyani river areas of the Zambesi Valley in a survey into the incidence of malaria, filariasis and trypanosomiasis.

(b) Randomly selected men, women and children from villages in the Kariba area of the Zambesi Valley were bled in a survey of filariasis.

(c) Staff and patients — mainly healthy women in maternity wards — at the Regina Coeli Mission Hospital in the Nyamaropa tribal trust lands beyond Inyanga, were bled following a report of typhoid fever in the area.

Lepers

Mr. B. P. B. Ellis, Leprosy Control Officer of the Ministry of Health, supplied sera from cases of leprosy additional to those included in the pilot survey. Patients came from a variety of tribal groups — principally Manyika and Ndau from Manicaland, Mazezuru and Kore Kore from Mashonaland and Tonga from the Binga area.

Patients with liver disease

Sera were obtained from 32 African patients at Harari Hospital and from three Europeans. The clinical diagnoses are presented with the results in Table IV.

Australia antigen tests

All sera were examined for Aul by the crossed-over immunoelectrophoretic method detailed previously (Cruickshank and Swanepoel, 1971) using anti-Aul serum prepared in baboons at the Natal Institute of Immunology.

RESULTS

The results are presented in tables I—IV.

Table I

THE INCIDENCE OF AUI IN URBAN BLOOD DONORS

Origin	No. examined	No. positive	% positive
(a) African donors in Salisbury:			
Community centres and clinics	101	1	1,0
Educational institutions	1 006	27	2,7
Prisons	1 731	55	3,2
Industry	761	32	4,2
Forces (Police and Air Force)	387	28	7,2
	3 986	143	Av. 3,6
(b) European donors in Salisbury	1 275	2	0,2
(c) African donors in Bulawayo	228	10	4,4

Table II

THE INCIDENCE OF AUI IN RURAL AFRICAN POPULATIONS

Origin	No. examined	No. positive	% positive
Regina Coeli Mission, Inyanga	71	0	—
Chitsungo, Zambezi Valley	652	10	1,5
Chitsunga, Zambezi Valley	169	9	4,7
Kariba, Zambezi Valley	144	8	5,6
	1 036	26	Av. 2,5

Table III

THE INCIDENCE OF AUI IN LEPERS

Type	No. examined	No. positive	% positive
Lepromatous leprosy	85	1	1,2
Tuberculoid leprosy	101	1	1,0
	186	2	Av. 1,1

Table IV

THE INCIDENCE OF AUI IN PATIENTS WITH LIVER DISEASE

Clinical diagnosis	No. examined	No. positive	% positive
Serum hepatitis	1	1	100
Infectious hepatitis	22	8	36,3
Undiagnosed jaundice	10	1	10
Hepatoma	1	1	100
Ca pancreas and liver failure	1	0	

Total: 6 746 specimens

DISCUSSION

The results of the present survey of the incidence of Aul in Rhodesia are best considered in relation to findings elsewhere. A selection of the surveys carried out over the last five years on apparently healthy populations are summarised in Table V. Most studies were performed on blood donors, but early work involving a search for a familial trait (Blumberg, Altar & Visnich, 1965) included more random population groups.

It is apparent that by and large the less sophisticated the population the higher is the incidence of Aul. The rate among Europeans in Rhodesia is comparable to the lowest in Europe and North America, while that among Africans is high by the standards of the developed countries.

Though some studies attempt to relate the antigen to certain genetic traits, the evidence that it is a transferable infectious agent is now irrefutable and the variations in incidence are probably best related to the environment and ways of

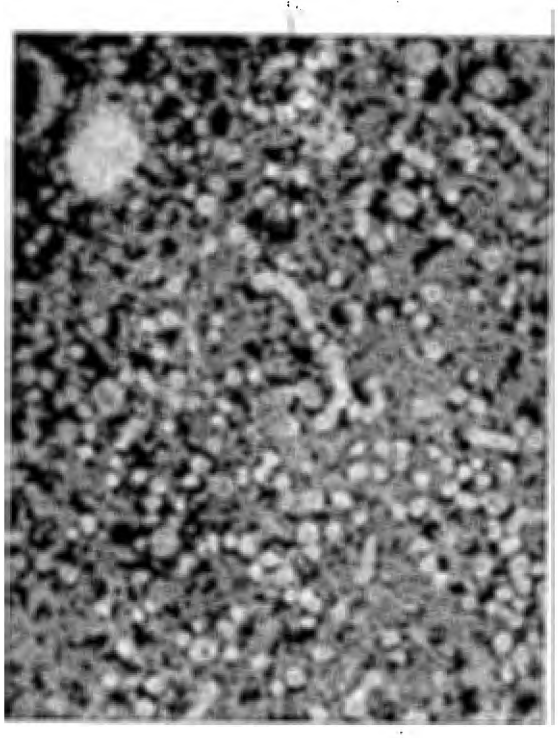

Fig. 1—Particles of Australia antigen from a Rhodesian blood donor. Serum was centrifuged at 105 000 xg for two hours and the resulting pellet resuspended in distilled water. An aliquot was transferred to a copper grid, stained with phosphotungstate ph 7,4 and examined in a Hitachi HU 11 electron microscope using a 50 kV voltage.

Table V

SURVEYS OF THE INCIDENCE OF AUI OVER THE LAST FIVE YEARS

Country	Source	% positive	Reference
EUROPE			
Lowest: Norway	Blood donors	0,16	Solaas, 1970
Highest: Greece	Blood donors	1,8	Blumberg, *et al*, 1970
N. AMERICA			
General	General population	0,1	Shulman, *et al*, 1970
New York	Blood donors	0,75	Gocke, *et al*, 1969
S. AMERICA			
Peru	Cashinahua Indians	20,2	Blumberg, *et al*, 1966
Mexico	Indians	0,4	Blumberg, *et al*, 1966
PACIFIC			
Australia	Aborigines	5,1	Blumberg, *et al*, 1966
New Guinea	Melanesians	3,6	Blumberg, *et al*, 1966
Bora Bora	Polynesians	2,5	Blumberg, *et al*, 1966
ASIA			
Japan	Blood donors (male)	1,23	Okochi & Murakami, 1968
Japan	Blood donors (female)	0,19	Okochi & Murakami, 1968
Phillipines	Filipinos	4,7	Blumberg, *et al*, 1970
Israel	General population	1,2	Blumberg, *et al*, 1970
AFRICA			
Tanzania	General population	0,8	Blumberg, *et al*, 1970
Ghana	General population	9,5	Blumberg, *et al*, 1970
Kenya	Blood donors	6,0	Bagshawe, *et al*, 1971
Natal	Blood donors	3,4	Grobbelaar, 1970
Rhodesia	Blood donors (African)	3,6	This survey
Rhodesia	Blood donors (European)	0,2	This survey

life of the different groups. Opportunities in tribal life for parental transfer of the agent include tribal markings and tattoos, circumcision and other minor surgical procedures undertaken without sterilisation of instruments. The uneven distribution of the antigen in rural populations suggests that careful epidemiological investigation might uncover parenteral transfer mechanisms of this nature at work in villages.

Urbanisation presents its own hazards and certain batches of donors have been found to have a strikingly high incidence of the antigen suggestive of small epidemics. This occurs in the young adult age group known to have the highest attack rate of hepatitis and involves particularly highly institutionalised groups such as those in the forces or training colleges and prisons. Accepting now that serum hepatitis is transmissible occasionally by the faecal-oral route (as by food handlers), common factors in each group include the sharing of bath and toilet facilities and even perhaps razors. It is most unlikely in the age of disposable syringes and needles that official inoculation programmes in such groups play a role in disseminating the antigen, but one throw-off of the disposable era is that it facilitates the work of the do-it-yourself medicine man. In two urban cases of Aul-positive hepatitis clear histories were obtained of repeated injections performed by unqualified persons known to be in the habit of treating people (Dr. T. Stamps, personal communication).

Since people in the survey were apparently well, it must be assumed that Aul-positive donors were in the throes of low-grade hepatitis or were carriers following frank or low-grade hepatitis. It is estimated that anicteric hepatitis occurs in 99 percent. of antigen positive infections (Hampers, Prager & Senior, 1964) and, therefore, most people who become carriers are probably unaware of antecedent disease. Long term carriers following hepatitis are reported to occur in the order of seven per cent. of cases (Wright, *et al.*, 1969). One apparently healthy donor in this survey continues to carry the antigen after four months, and a patient remains positive a year after an attack of hepatitis. This patient's liver function tests are still abnormal (Dr. E. Taube and Dr. K. G. Gadd, personal communication).

The risk of hepatitis following transfusion with Aul-positive blood is difficult to assess as the

majority of cases are anicteric. Out of 40 persons who were transfused with Au1-positive blood, Gocke & Kavey (1969), found that 70 per cent. developed hepatitis, while only four out of 69 transfused with Au I-negative blood acquired the disease. Thus, though eliminating Au1-positive blood, by no means eliminates post-transfusion hepatitis, it is estimated that this procedure will reduce the incidence by 37 per cent. or more (Chalmers and Alter, 1971). The case of serum hepatitis reported in this paper is the second post-transfusion fatality recorded this year in Rhodesia. In both instances Au1 was present in high concentration in the serum of the recipient, but only in the case reported in the pilot survey (Cruickshank and Swanepoel, 1971) was the antigen detected in a donor.

On the basis of the present survey, replacing Au1-positive blood adds four per cent. to the total cost of African blood in Rhodesia. It may be possible to reduce this figure by eliminating known positive donors, but the high rate of turnover of the donor population may militate against it. Only the continued testing of Salisbury donors will show whether or not any appreciable reduction can be achieved.

Pressure is now being brought to screen food handlers, medical personnel and others who may, in the course of their work, be in a position to transmit the agent, so that those who turn out to be positive may be found safer work during the time they remain potentially infectious (Chalmers & Alter, 1971). Further, in their own interests, carriers should undergo liver function tests to detect any active hepatitis. A recent study reported finding evidence of acute or chronic hepatitis by liver biopsy in 22 out of 25 Au1 antigen carriers (Singleton, *et al.*, 1971).

The findings in the lepers and in the hepatitis cases reinforce those published earlier that lepers here unlike those in the Antipodes do not have a high incidence of Au1 carriage and that many cases diagnosed as infectious hepatitis are in fact of the more serious serum variety.

SUMMARY

Australia antigen was found in the sera of 3,6 per cent of African blood donors from Salisbury and in 4,4 per cent. from Bulawayo and in 0,2 per cent. of European donors in Salisbury.

The incidence in rural African groups varied from nil to 5,6 per cent.

The epidemiology and significance of these findings are discussed.

The low incidence among lepers in Rhodesia is confirmed.

REFERENCES

BAGSHAWE, A. F., PARKER, A. M. & JINDANI, A. (1971). *Brit. med. J.*, **i**, 88.
BLUMBERG, B. S., ALTER, H. J. & VISNICH, S. (1965). *J. Amer. med. Assoc.*, **191**, 541.
BLUMBERG, B. S., MELARTIN, L., GUINTO, R. A. & WERNER, B. (1966). *Amer. J. Hum. Genet.*, **18**, 594.
BLUMBERG, B. S., SUTNICK, A. I. & LONDON, W. T. (1970) *Amer. J. Med.*, **48**, 1.
CRUICKSHANK, J. G. & SWANEPOEL, R. (1970). *C. Afr.J. Med.*, **17**, 205.
GOCKE, D. J. & KAVEY, N. B. (1969). *Lancet* **i**, 1056.
GOCKE, D. J., GREENBERG, H. B. & KAVEY, N. B. (1969). *Lancet* **ii** 248.
GROBBELAAR, B. G. (1970) *in* Ann. Rep. of Natal Inst. for Immunology.
HAMPERS, C. L., PRAGER, D. & SENIOR, J. R. (1964). *New Eng. J. Med.*, **271**, 747.
OKOCHI, K. & MURAKAMI, S. (1968). *Vox Sang.*, **15**, 374.
SINGLETON, J. W., FITCH, R. A., MERRILL, D. A., KOHLER, P. F. & RETTBERG, W. A. H. (1971). *Lancet* **ii**, 785.
SOLAAS, M. H. (1970). *Scand. J. Haematol.*, **7**, 506.
SHULMAN, N. R. (1970). *Amer. J. Med.* **49**, 669.
SHULMAN, N. R., HIRSCHMAN, R. J. & BARKER, L. (1970). *Ann. Int. Med.* **72**, 257.
WRIGHT, R., MCCOLLUM, R. W. & KLATSKIN, G. (1969). *Lancet* **ii**, 117.

Acknowledgments

We are grateful to the Secretary for Health for permission to publish certain of the data.